

Abstracts

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Intra-operative colon mucosal oxygen saturation during aortic surgery
Lee ES, Bass A, Arko FR, et al. J Surg Research 2006;136:19-24.

Conclusion: A measurement of colonic mucosal oxygen saturation correlates with the stoppage and restoration of aortic flow during aortic surgery.

Summary: Colon ischemia complicating aortic reconstruction has a high mortality rate. The authors developed a method of measuring colonic mucosal ischemia during aortic surgery that correlates with intraoperative events. A spectrophotometer probe is inserted into a patient's rectum before aortic reconstruction. The device allows continuous measurement of colonic mucosal oxygen saturation (CMOS). It was tested in 25 patients undergoing aortic reconstruction. Buccal mucosal oxygen saturation, mean arterial pressure, heart rate, pulse oximetry, and arrest and restoration of flow in the aorta were also recorded during each operative procedure.

An endovascular aneurysm repair (EVAR) was performed in 20 patients and an open repair was performed in five patients. During EVAR, CMOS decreased from a baseline of $56\% \pm 8\%$ to $26\% \pm 17\%$ during the period of infrarenal aortic balloon occlusion ($P < .0001$). During open repair, CMOS also decreased from $56\% \pm 9\%$ to $15\% \pm 19\%$ during clamping of the infrarenal aorta and iliac arteries ($P < .0001$). With restoration of aortic flow in both open procedures and EVAR, CMOS returned to baseline values ($56.5\% \pm 10\%$, $P = .81$ compared with baseline). The time for CMOS to recover after restoration of aortic flow was 6.4 ± 3.3 minutes.

Comment: The technique offers a potential method of monitoring colonic perfusion intraoperatively. Obviously, a number of details still need to be determined, such as what is the lowest level of colonic mucosal oxygen saturation that will sustain colonic viability. In addition, the potential ability of this technique to monitor for postoperative colonic ischemia seems like an interesting line of inquiry.

Clinical syndromes and clinical outcome in patients with pulmonary embolism: Findings from the RIETE Registry

Lobo JL, Zorrilla V, Aizpuru F, et al. Chest 2006;130:1817-22.

Conclusion: Patients with pulmonary embolism (PE) who suffer pulmonary infarction as a manifestation of their PE have a better initial and long-term prognosis than patients with a PE that presents as dyspnea or circulatory collapse.

Summary: Information in this study was derived from the Registro Informático de la Enfermedad Tromboembólica (RIETE) Registry of consecutive patients with acute venous thromboembolism (VTE). This is a multi-center observational registry that gathers and analyses data of clinical outcomes and treatment patterns in patients with symptomatic, objectively confirmed, acute VTE (deep venous thrombosis or PE). The current study focused on patients enrolled in the registry with PE who did not have pre-existing cardiac or pulmonary disease. Patients were classified into three clinical syndromes: pulmonary infarction, dyspnea, or circulatory collapse. Each group was analyzed with respect to clinical characteristics, laboratory findings, and 3-month outcomes.

There were 3391 patients (82% of the RIETE Registry) who presented with acute, symptomatic, objectively confirmed PE and who did not have chronic lung disease or heart failure. Of these patients, 1709 (50%) had pulmonary infarction, 1083 (32%) had isolated dyspnea, and 599 (18%) presented with circulatory collapse. Of the entire group, 149 (4.4%) died during the first 15 days after presentation, and 2.5% of the patients with pulmonary infarction died between day 16 and day 90. Of those with isolated dyspnea, 6.2% died (odds ratio [OR], 2.6; 95% confidence interval [CI] 1.7 to 3.8) and 6.5% with circulatory collapse died (OR 2.7, 95% CI, 1.7 to 4.2). Thirty-one patients had recurrent PE. Fourteen of these patients had initially presented with pulmonary infarction, and five died (36%). Ten of the patients had initially presented with dyspnea and five died (50%). All seven patients with recurrent PE who initially presented with circulatory collapse died of their second PE.

Comment: The data make sense. Patients with pulmonary infarction usually present with pleuritic chest pain or hemoptysis reflecting peripheral emboli. Those with dyspnea and circulatory collapse have more central and larger emboli and thus have more extensive vascular occlusion. It is important to note that, no matter the initial presentation, recurrent PE ≤ 90 days is a highly fatal event.

Preoperative MDCT evaluation of the artery of Adamkiewicz and its origin

Takase K, Akasaka J, Sawamura Y, et al. J Comput Assist Tomogr 2006;30:716-22.

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Conclusion: Multidetector computed tomography (MDCT) allows identification of the entire length of the artery of Adamkiewicz and its origin in patients with thoracoabdominal aneurysms.

Summary: The artery of Adamkiewicz supplies the lower one third of the spinal cord and can arise from a posterior branch of an intercostal or lumbar artery. Identification of the artery of Adamkiewicz can be crucial in planning which, if any, intercostal vessels should be reimplanted or preserved during thoracoabdominal aneurysm repair. Proper identification and preservation of the artery of Adamkiewicz may reduce neurologic complications of thoracoabdominal aneurysm repair. This applies to both open and endovascular repair.

In this study, the authors evaluated the capability of an eight-slice multidetector CT scanner with 1-mm collimation, along with a rapid injection protocol, to evaluate the continuity of the artery of Adamkiewicz and its parent artery. Ten patients with thoracoabdominal vascular disease underwent MDCT, and in nine patients, the artery of Adamkiewicz was clearly visualized. The entire length of the intercostal or lumbar arteries leading to the artery of Adamkiewicz could be traced in eight patients. Three patients had occlusion of the parent artery of the artery of Adamkiewicz.

Comment: It makes sense that identification of a patent artery of Adamkiewicz and its parent vessel may be important in planning thoracoabdominal aneurysm repair. This may allow targeted preservation or reattachment of specific intercostal or lumbar vessels. It may also be equally important to know which patients have occluded parent vessels to the artery of Adamkiewicz. Perhaps in such cases, reattachment or preservation of intercostal lumbar vessels is not necessary in thoracoabdominal aneurysm repair. Equally possible, however, is that reattachment of additional vessels to preserve collateral flow will be required.

The pathogenesis of venous thromboembolism: Evidence for multiple interrelated causes

Brouwer JL, Veeger NJ, Kluin-Nelemans HC, et al. Ann Intern Med 2006;145:807-15.

Conclusion: Multiple genetic and environmental risk factors are additive in contributing to the risk of venous thromboembolism (VTE).

Summary: The authors sought to assess the effects of multiple thrombophilic defects and risk factors with respect to the absolute risk for VTE. This was a retrospective family cohort study conducted in a single university hospital in the Netherlands. Study subjects were 468 relatives of 91 probands with symptomatic hereditary deficiencies of protein C, protein S, or antithrombin. Relatives were tested for 10 thrombophilic defects in addition to the index deficiency. They were also assessed for exogenous risk factors such as immobilization, trauma, surgery, oral contraceptives, and pregnancy. Annual incidence and relative risk for VTE were compared in subjects with deficiencies of protein C, protein S, or antithrombin and in nondeficient relatives.

The annual incidence of VTE per 100 patient years in relatives with 0, 1, 2, or more additional thrombophilic defects or deficiencies were 1.16 (95% confidence interval [CI], 0.6 to 2.3), 1.75 (95% CI, 1.17 to 2.53), and 2.64 (95% CI, 1.67 to 3.96). The relative risk in nondeficient relatives was 0.06 (95% CI, 0.002 to 0.33) per 100 patient years. Adjusted relative risks were 16.3 (95% CI, 2.0 to 131.0), 50.3 (95% CI, 6.5 to 390), and 103 (95% CI, 12.5 to 843). In proband relatives with deficiencies of protein C, protein S, or antithrombin, 38% with no additional defects, 57% with one additional defect, and 81% with two or more additional defects had VTE by the age of 65 years. In comparison, only 5% of nondeficient relatives had VTE at 65 years ($P < .001$). In relatives with deficiencies of protein S, protein C, or antithrombin, and additional thrombophilic deficiencies or defects, exogenous risk factors further increased the risk of VTE from 1.2% to 2.5% per year (relative risk, 2.1; 95% CI, 1.1 to 4.2).

Comment: The basic message is simple. In individuals who already have a thrombophilic defect (protein C, protein S, or antithrombin deficiencies in this study), the risk of VTE increases further with additional thrombophilic defects or environmental risk factors. This, of course, is not the first time such an observation has been made. The magnitude of increased risk for relatives of probands with symptomatic protein C, protein S, or antithrombin deficiency is, however, sufficiently great that one wonders if prophylactic anticoagulation may be indicated in such patients.

Familial cervical artery dissections: Clinical, morphologic and genetic studies

Martin JJ, Hausser I, Lyrer P, et al. Stroke 2006;37:2924-9.

Conclusion: Familial patients with spontaneous cervical carotid artery dissection are at high risk for recurrent or multiple cervical carotid artery dissections.

Summary: Only a minority of patients with spontaneous carotid artery dissection have a clinical diagnosis of a connective tissue disorder. However, dermatologic biopsy specimens in young patients with spontaneous carotid artery dissection have shown abnormalities in up to 50% to 60%, despite the absence of clinical signs of connective tissue disorder (Neurology 2001; 57:24-30).

The authors identified seven families with 15 patients with spontaneous carotid artery dissection. Patients were examined by a dermatologist, a neuroradiologist, and a neurologist for clinical characteristics of an underlying connective tissue disorder. Eleven patients had a skin biopsy. Coding sequences COL3A1, COL5A1, COL5A2, and part of COL1A1 were also examined.

The mean age at the time of the first spontaneous carotid artery dissection was 36.2 years (median, 32 years; range, 18 to 59 years). There were nine men and six women, and two patients had bilateral spontaneous carotid dissection. One patient had five dissections within an 8-year period. There was a high correlation among families between affected vessels (ie, vertebral or carotid arteries, or both) and between age at the first dissection. All but one patient analyzed with skin biopsies showed normal connective tissue morphology. One patient had mild findings consistent with vascular Ehlers-Danlos syndrome type 4. Four patients were identified with mutations in COL genes.

Comment: Patients with familial spontaneous carotid artery dissection are younger than the general cohort of patients with spontaneous carotid artery dissection. Microscopic analysis of skin biopsy specimens is essentially unrevealing. Younger patients with spontaneous carotid artery dissection should perhaps be counseled about a familial tendency for this disease, but it does not appear that genetic testing, at least as we currently understand it, is helpful.

Long-term survival in patients presenting with type B acute aortic dissection: Insights from the International Registry of Acute Aortic Dissection

Tsai TT, Fattori R, Trimarchi S, et al; and the International Registry of Acute Aortic Dissection (IRAD). Circulation 2006;114:2226-31.

Conclusion: There is a high mortality rate in patients with acute type B aortic dissection who survive to hospital discharge, with almost one in four patients dead at 3 years.

Summary: Patients with acute type B aortic dissection generally do well in the hospital, with 90% surviving to hospital discharge with effective antihypertensive therapy (JAMA 2000;283:897-903). Late follow-up of such patients has not been well studied and has been primarily restricted to single-center reports. The authors used data from a contemporary registry of acute type B aortic dissections to analyze long-term survival in such patients.

Data were derived from the International Registry of Acute Aortic Dissection (IRAD). Patients were accumulated from 1996 to 2003. The basis of this study was 242 consecutive patients discharged from the hospital alive with acute type B aortic dissection. Survival was determined with Kaplan-Meier survival curves. Cox proportional hazards analysis was used to identify predictors of mortality during follow-up.

Three-year survival for patients treated medically was $77.6\% \pm 6.1\%$, $82.8\% \pm 18.9\%$ for those treated surgically, and $76.2\% \pm 25.2\%$ (log rank $P = .61$) for those treated with endovascular therapy. Predictors of mortality during follow-up included a history of aortic aneurysm (hazard ratio [HR], 2.17; 95% confidence interval [CI], 1.03 to 4.59; $P = .04$), history of atherosclerosis (HR 2.41; 95% CI 1.32 to 4.66; $P < .01$), plural effusion (HR, 2.56; 95% CI, 1.18 to 5.58; $P = .02$), hypotension and shock in the hospital (HR, 12.5; 95% CI, 3.21 to 48.21; $P < .01$), and female gender (HR, 1.99; 95% CI, 1.07 to 3.71).

Comment: The predictors of mortality after acute type B aortic dissection identified in this report have all been previously identified. No differences in survival existed between medically managed, surgically managed, and endovascularly managed patients. This was not a randomized study, however, and it is possible that the patients treated with either surgical or endovascular techniques had additional risk factors for long-term mortality. The bulk of the data, however, do suggest that it is going to be difficult to acquire sufficient patients with sufficient power to test the hypothesis of whether immediate endovascular therapy of acute type B dissection provides better long-term results than standard medical management and intervention-only for complications of dissection or failure of medical management.

Sonographic elasticity imaging of acute and chronic deep venous thrombosis in humans

Rubin JM, Xie H, Kim K, et al. J Ultrasound Med 2006;25:1179-86.

Conclusion: Ultrasound-based elasticity imaging may help distinguish chronic from acute thrombosis and thus be useful in the clinical evaluation of patients with post-thrombotic syndrome.

Summary: Distinguishing acute from chronic venous thrombosis is difficult with ultrasound B-mode imaging alone but may be important in a patient with a history of venous thrombosis and new symptoms in the leg. Under such circumstances, determining the age of the thrombus has significant clinical implications. One method of potentially aging venous thrombus is sonographic elasticity imaging. This technique allows direct assessment of tissue hardness. Because deep vein thrombi harden as they age, the technique may be possible to use in distinguishing acute from chronic venous thrombus.

The authors of this study analyzed data in 46 patients: 23 had acute venous thrombi (mean age, 5.7 days) and 23 had chronic venous thrombi (>8 months). A 5-MHz linear array transducer was used to subject each thrombus to free-hand external deformation. Strains in the thrombi were normalized to average strain between the skin surface and the back wall of the vein. Thrombus echogenicity was measured by comparing the adjacent arterial lumen with the echogenicity of the thrombus.

The acute cases had a median normalized strain magnitude of 2.75 (interquartile range, 2.4 to 3.71). In chronic cases, the mean normalized strain magnitude was 0.94 (interquartile range, 0.48 to 1.36, $P < 10^{-7}$). Echogenicity differences between the populations were also highly significant ($P < 10^{-8}$).

Comment: Reliably determining the age of a venous thrombus is one of the Holy Grails of noninvasive vascular testing. The population studied here, however, was somewhat artificial compared with the clinical situation. The current population consisted of patients with known acute vs known chronic thrombi. Sonographic elasticity imaging needs to be evaluated in patients with a combination of acute and chronic venous thrombi to more mimic the clinical question of determining thrombus age in a patient with acute symptoms and known previous venous thrombosis.

Hypertension accelerated experimental abdominal aortic aneurysm through upregulation of nuclear factor κ B and Ets

Shiraya S, Miwa K, Aoki M, et al. Hypertension 2006;48:628-36.

Conclusion: Hypertension accelerates progression of experimental abdominal aortic aneurysms (AAAs) by upregulation of Ets and nuclear factor κ B (NF κ B).

Summary: The pathogenesis of AAA is complex but appears to involve upregulation of certain matrix metalloproteinases (MMPs). Experimental AAAs are characterized by upregulation of transcription factors, NF κ B, which regulate transcription, and Ets, which regulates expression of MMPs. The role of hypertension in pathogenesis of AAA is controversial, with some studies suggesting it contributes to aneurysm formation and other studies suggesting it is not an independent risk factor for aneurysm formation.

In this study, the authors used an experimental model of AAA to investigate the effects of hypertension on transcription factors NF κ B and Ets. They also investigated the effects of hypertension on progression of AAA. Experimental AAAs were produced by elastase perfusion in both normotensive and hypertensive rats. Compared with normotensive rats, the size of the AAAs increased more rapidly in the hypertensive rats. Western blot analysis indicated the expression of MMP-2, MMP-3, MMP-9, and MMP-12, as well as that of intercellular adhesion molecule, was increased in hypertensive rats. This was accompanied by upregulation of NF κ B and Ets. There was also increased activity of MMPs in the aortas of hypertensive rats vs those of normotensive rats.

Chimeric decoy oligodeoxynucleotide (ODN) inhibits expression of NF κ B and Ets. Transfection of the ODN resulted in significant inhibition of aortic dilatation in both hypertensive and normotensive rats. Transfection with chimeric decoy ODN resulted in significant inhibition of degeneration of elastic fibers in aortas of both normotensive and hypertensive rats. Expression of MMP-2, MMP-3, MMP-9, and MMP-12 and intercellular adhesion molecule was decreased by chimeric decoy ODN. This was accompanied by inhibition of macrophage migration.

Comment: The study indicates hypertension accelerates progression of AAA in an experimental rat model. Perhaps inhibition of NF κ B and Ets may someday be used as a method of decreasing aneurysm expansion in both normotensive and hypertensive patients.